L Number	Hits	Search Text	DB	Time stamp
1	1815	"androgen receptor"	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/01/08 13:46
2	13	"coregulatory protein"	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/01/08 13:47
3	11222	"transcription factor"	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/01/08 13:47
4	83	"androgen receptor" SAME "transcription factor"	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/01/08 13:47
5	689	inhibitor SAME androgen	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/01/08 13:49
6	0	(inhibitor SAME androgen) and "method of screening"	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/01/08 13:48
7	12	"screening inhibitors" and "androgen receptor"	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/01/08 14:30
8	825	antiandrogen	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/01/08 13:51
9	0	antiandrogen and "method of screening"	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/01/08 13:52
10	520	"transcription factor" and "androgen receptor"	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/01/08 14:32
11	129	("transcription factor" and "androgen receptor") and "protein interaction"	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/01/08 14:33

1 0 US 20040006016 A1 US-PGPUB 20040108 245 Novel 27875, 22025 ,27420, 17906, 16319, 55092 and 10218 molecules and uses therefor 514/12 435/183; 435/320.1; 435/325; 435/6; 435/69.1; 530/350; 536/23.2 Kapeller-Libermann, Rosana et al. 0 0 0 0 0 0 0 PGPubs Full Image US 20040006016 0

1 0 US 20040005700 A1 US-PGPUB 20040108 Poroplasts 435/317.1 435/252.3; 435/455 Surber, Mark W. et al. 0 0 0 0 0 0 0 Default 0

1 0 US 20040005563 A1 US-PGPUB 20040108 Methods of diagnosis of ovarian cancer, compositions and methods of screening for modulators of ovarian cancer 435/6 435/183; 435/320.1; 435/366; 435/69.1; 435/7.23; 536/23.2 Mack, David H. et al. 0 0 0 0 0 0 0 Default 0

1 0 US 20040005560 A1 US-PGPUB 20040108 165 Novel full-length cDNA 435/6 435/183; 435/320.1; 435/325; 435/69.1; 530/350; 530/388.1; 536/23.5 Isogai, Takao et al. 0 0 0 0 0 0 PGPubs Full Image US 20040005560 0

1 0 US 20040003418 A1 US-PGPUB 20040101 Nucleic acid and corresponding protein entitled 158P3D2 useful in treatment and detection of cancer 800/3 424/146.1; 435/326; 514/12; 514/44; 800/8 Jakobovits, Aya et al. 0 0 0 0 0 0 0 Default 0

1 0 US 20030235860 A1 US-PGPUB 20031225 Interactions between AR, ER, TR2, and TR4 435/7.1 514/12 Chang, Chawnshang 0 0 0 0 0 0 0 Default 0

1 0 US 20030232335 A1 US-PGPUB 20031218 Minicell-based screening for compounds and proteins that modulate the activity of signalling proteins 435/6 435/7.1; 435/7.2 Surber, Mark W. et al. 0 0 0 0 0 0 0 Default 0

1 0 US 20030229904 A1 US-PGPUB 20031211 Nucleic acid and corresponding protein entitled 161P5C5 useful in treatment and detection of cancer 800/6 424/146.1; 435/6; 435/7.21; 514/44; 530/387.2 Challita-Eid, Pia M. et al. 0 0 0 0 0 0 0 Default 0

1 0 US 20030228607 A1 US-PGPUB 20031211 Screening method and modulators having an improved therapeutic profile 435/6 435/7.2; 530/358 Wagner, Brandee Lynn et al. 0 0 0 0 0 0 0 Default 0

1 0 US 20030224444 A1 US-PGPUB 20031204 Antibodies to native conformations of membrane proteins 435/7.1 435/326; 435/69.1; 530/387.1 Sabbadini, Roger A. et al. 0 0 0 0 0 0 0 Default 0

1 0 US 20030224390 A1 US-PGPUB 20031204 Method of identifying conformation-sensitive binding peptides and uses thereof

435/6 Fowlkes, Dana M. et al. 0 0 0 0
0 0 0 Default 0
1 0 US 20030224369 A1 US-PGPUB 20031204
Reverse screening and target identification with minicells 435/6
Surber, Mark W. et al. 0 0 0 0 0 0 0 Default
0
1 0 US 20030223997 A1 US-PGPUB 20031204
Nucleic acid and corresponding protein entitled 121P1F1 useful in treatment and detection of cancer 424/155.1 435/226; 435/320.1; 435/325; 435/6; 435/69.1; 530/388.8; 536/23.2 Challita-Eid, Pia M. et al. 0 0
0 0 0 0 0 Default 0
1 0 US 20030223990 A1 US-PGPUB 20031204
Nucleic acid and corresponding protein entitled 193P1E1B useful in treatment and detection of cancer 424/141.1 424/146.1; 435/338; 530/388.15; 530/388.26; 800/6 Raitano, Arthur B. et al. 0 0 0 0
0 0 0 Default 0
1 0 US 20030219888 A1 US-PGPUB 20031127
Minicell-based bioremediation 435/262.5 Segall, Anca M. et al. 0 0 0 0 0 0 0 Default 0
1 0 US 20030219806 A1 US-PGPUB 20031127 Novel 18607, 15603, 69318, 12303, 48000, 52920, 5433, 38554, 57301, 58324, 55063, 52991, 59914, 59921 and 33751 molecules and uses therefor 435/6 435/183; 435/320.1; 435/325; 435/69.1; 514/12; 530/350; 530/388.1; 536/23.2
Glucksmann, Maria A. et al. 0 0 0 0 0 0 0
Default 0
1 0 US 20030219789 A1 US-PGPUB 20031127
36P6D5: secreted tumor antigen 435/6 435/7.23
Raitano, Arthur B. et al. 0 0 0 0 0 0 0
Default 0
1 0 US 20030219767 A1 US-PGPUB 20031127
Compositions, kits, and methods for identification, assessment, prevention, and therapy of breast cancer 435/6 435/7.23 Ayers, Mark D. et al.
0 0 0 0 0 0 Default 0
1 0 US 20030219766 A1 US-PGPUB 20031127
103P2D6: tissue specific protein highly expressed in various cancers
435/6 435/320.1; 435/325; 435/69.1; 435/7.23; 530/350; 530/388.8; 536/23.5; 800/8 Raitano, Arthur B. et al. 0 0 0 0
0 0 0 Default 0
1 0 US 20030219738 A1 US-PGPUB 20031127
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Challita-Eid, Pia M. et al. 0 0 0 0 0 0 0
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1 0 US 20030219444 A1 US-PGPUB 20031127
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530/391.1; 536/23.53; 800/8 Faris, Mary et al. 0 0 0 0
0 0 0 Default 0
1 0 US 20030219408 A1 US-PGPUB 20031127
Methods of making pharmaceutical compositions with minicells
424/93.2 Sabbadini, Roger A. et al. 0 0 0
0 0 0 0 Default 0
1 0 US 20030215852 A1 US-PGPUB 20031120 Novel
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Mark, Robert John et al. 0 0 0 0 0 0 0
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1 0 US 20030215829 A1 US-PGPUB 20031120
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0 0 0 Default 0
1 0 US 20030215449 A1 US-PGPUB 20031120
Proteins and nucleic acids encoding same 424/146.1 435/7.23
Mezes, Peter D. et al. 0 0 0 0 0 0 0 Default
0
1 0 US 20030213004 A1 US-PGPUB 20031113
Nucleic acids and corresponding proteins entitled 101P3A11 or PHOR-1 useful in
treatment and detection of cancer 800/8 424/146.1; 435/183; 435/320.1;
435/325; 435/326; 514/44; 530/388.26; 536/23.2 Jakobovits, Aya et al. 0
0 0 0 0 0 0 Default 0
1 0 US 20030211599 A1 US-PGPUB 20031113
Minicell-based delivery agents 435/325 435/252.3
Sabbadini, Roger A. et al. 0 0 0 0 0 0 0
Default 0
1 0 US 20030211086 A1 US-PGPUB 20031113
Minicell-based selective absorption 424/93.21 424/1.49; 424/1.73;
435/325 Berkley, Neil et al. 0 0 0 0 0 0
0 Default 0
1 0 US 20030208039 A1 US-PGPUB 20031106 Novel
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Padigaru, Muralidhara et al. 0 0 0 0 0 0 0
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1 0 US 20030207835 A1 US-PGPUB 20031106
Nucleic acid and corresponding protein named 158P1D7 useful in the treatment
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Faris, Mary et al. 0 0 0 0 0 0 0 Default
0
1 0 US 20030207833 A1 US-PGPUB 20031106
Pharmaceutical compositions with minicells 514/44 424/93.21
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0

1 0 US 20030206905 A1 US-PGPUB 20031106
Nucleic acid and corresponding protein entitled 161P2F10B useful in treatment and detection of cancer 424/145.1 435/326; 530/388.1; 800/6
Jakobovits, Aya et al. 0 0 0 0 0 0 0 Default 0
1 0 US 20030203481 A1 US-PGPUB 20031030
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0 0 0 0 0 0 0 Default 0
1 0 US 20030203411 A1 US-PGPUB 20031030
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Sabbadini, Roger A. et al. 0 0 0 0 0 0 0
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Minicell-based diagnostics 424/1.49 424/9.34; 424/9.5
Sabbadini, Roger A. et al. 0 0 0 0 0 0 0
Default 0
1 0 US 20030199470 A1 US-PGPUB 20031023
Nucleic acid and corresponding protein named 158P1D7 useful in the treatment and detection of bladder and other cancers 514/44 424/155.1; 435/6; 435/7.23; 514/12 Faris, Mary et al. 0 0 0 0 0 0 0
Default 0
1 0 US 20030199089 A1 US-PGPUB 20031023
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1 0 US 20030199088 A1 US-PGPUB 20031023
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1 0 US 20030199005 A1 US-PGPUB 20031023 Solid supports with minicells 435/7.21 435/325 Sabbadini, Roger et al. 0 0 0 0 0 0 Default 0
1 0 US 20030198996 A1 US-PGPUB 20031023
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1 0 US 20030198995 A1 US-PGPUB 20031023
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Default 0
1 0 US 20030198990 A1 US-PGPUB 20031023
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0 0 0 0 0 Default 0
1 0 US 20030194798 A1 US-PGPUB 20031016
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Surber, Mark W. et al.0 0 0 0 0 0 0 Default
0
1 0 US 20030194714 A1 US-PGPUB 20031016
Minicell-based transformation 435/6 435/325; 435/455
Sabbadini, Roger A. et al. 0 0 0 0 0 0 0
Default 0
1 0 US 20030194407 A1 US-PGPUB 20031016
103P2D6: tissue specific protein highly expressed in various cancers
424/155.1 435/196; 435/320.1; 435/325; 435/338; 435/6; 435/69.1;
435/7.23; 530/388.26; 536/23.2; 800/8 Raitano, Arthur B. et al. 0
0 0 0 0 0 0 Default 0
1 0 US 20030191073 A1 US-PGPUB 20031009
Nucleic acid and corresponding protein entitled 161P2F10B useful in treatment
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Challita-Eid, Pia M. et al. 0 0 0 0 0 0 0
Default 0
1 0 US 20030190749 A1 US-PGPUB 20031009
Minicell-producing parent cells 435/375 Surber,
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1 0 US 20030190683 A1 US-PGPUB 20031009
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Sabbadini, Roger A. et al. 0 0 0 0 0 0 0
Default 0
1 0 US 20030190601 A1 US-PGPUB 20031009 Target
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Roger A. et al. 0 0 0 0 0 0 0 Default 0
1 0 US 20030186863 A1 US-PGPUB 20031002 Nck
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530/326; 530/327 Sparks, Andrew B. et al. 0 0 0 0
0 0 0 Default 0
1 0 US 20030186385 A1 US-PGPUB 20031002
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Koide, Shohei 0 0 0 0 0 0 0 Default
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1 0 US 20030186273 A1 US-PGPUB 20031002 15603,
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0 0 Default 0
1 0 US 20030181692 A1 US-PGPUB 20030925 207
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0 Default 0
1 0 US 20030180947 A1 US-PGPUB 20030925
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Wu, J.H. David et al. 0 0 0 0 0 0 0 Default 0

1 0 US 20030175736 A1 US-PGPUB 20030918

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Chinnaiyan, Arul M. et al. 0 0 0 0 0 0 0 Default 0

1 0 US 20030170626 A1 US-PGPUB 20030911

Nucleic acid and corresponding protein entitled 85P1B3 useful in treatment and detection of cancer 435/6 424/155.1; 435/7.23 Raitano, Arthur B. et al. 0 0 0 0 0 0 Default 0

1 0 US 20030166850 A1 US-PGPUB 20030904

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Jones, Philip G. et al. 0 0 0 0 0 0 0 Default 0

1 0 US 20030166526 A1 US-PGPUB 20030904

Nucleic acid and corresponding protein named 158P1H4 useful in the treatment and detection of bladder and other cancers 514/12 424/146.1; 435/6; 514/44

Challita-Eid, Pia M. et al. 0 0 0 0 0 0 0 Default 0

1 0 US 20030166279 A1 US-PGPUB 20030904

Minicell-based transfection 435/449 435/320.1; 435/325

Sabbadini, Roger A. et al. 0 0 0 0 0 0 0 Default 0

1 0 US 20030166099 A1 US-PGPUB 20030904

Minicells comprising membrane proteins 435/69.1 435/325

Sabbadini, Roger A. et al. 0 0 0 0 0 0 0 Default 0

1 0 US 20030157597 A1 US-PGPUB 20030821

103P2D6: tissue specific protein highly expressed in various cancers

435/69.1 435/320.1; 435/325; 530/350; 536/23.5 Raitano, Arthur B. et al. 0 0 0 0 0 0 0 Default 0

1 0 US 20030149531 A1 US-PGPUB 20030807

Serpentine transmembrane antigens expressed in human cancers and uses thereof

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1 0 US 20030134784 A1 US-PGPUB 20030717

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1 0 US 20030124579 A1 US-PGPUB 20030703

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1 0 US 20030124530 A1 US-PGPUB 20030703
Sequence-directed DNA-binding molecules compositons and methods
435/6 Edwards, Cynthia A. et al. 0 0 0 0
0 0 0 Default 0
1 0 US 20030109683 A1 US-PGPUB 20030612
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O'Malley, Bert W. et al. 0 0 0 0 0 0 0
Default 0
1 0 US 20030108963 A1 US-PGPUB 20030612 Novel genes, compositions, kit, and methods for identification, assessment, prevention and therapy of prostate cancer 435/7.23 435/183; 435/320.1; 435/325; 435/69.3; 530/350; 530/388.26; 536/23.2 Schlegel, Robert et al. 0 0
0 0 0 0 0 Default 0
1 0 US 20030092119 A1 US-PGPUB 20030515
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0 0 0 0 Default 0
1 0 US 20030091569 A1 US-PGPUB 20030515
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0 0 Default 0
1 0 US 20030091562 A1 US-PGPUB 20030515
Nucleic acid and corresponding protein entitled 101P3A41 useful in treatment and detection of cancer 424/142.1 424/143.1; 424/146.1 Jakobovits, Aya et al. 0 0 0 0 0 0 Default 0
1 0 US 20030077800 A1 US-PGPUB 20030424 ARIP4 gene and protein 435/196 424/94.6; 536/23.2 Rouleau, Nathalie et al. 0 0 0 0 0 0 0 Default 0
1 0 US 20030077606 A1 US-PGPUB 20030424
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1 0 US 20030068672 A1 US-PGPUB 20030410 Mu opioid receptor methods 435/69.1 435/320.1; 435/325; 530/350; 536/23.5 Yu, Lei 0 0 0 0 0 0 0
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1 0 US 20030065004 A1 US-PGPUB 20030403
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Hutchinson, John H. et al. 0 0 0 0 0 0
0 Default 0
1 0 US 20030064418 A1 US-PGPUB 20030403
55P4H4: gene expressed in various cancers 435/7.23 530/324; 530/350 Faris, Mary et al. 0 0 0 0 0 0
0 Default 0

1 0 US 20030059895 A1 US-PGPUB 20030327
125P5C8: a tissue specific protein highly expressed in various cancers
435/69.3 435/325; 435/70.21; 530/350; 530/388.8; 800/8 Faris,
Mary et al. 0 0 0 0 0 0 0 Default 0
1 0 US 20030054438 A1 US-PGPUB 20030320
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1 0 US 20030032087 A1 US-PGPUB 20030213
121P1F1: a tissue specific protein highly expressed in various cancers
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536/23.2; 800/10 Challita-Eid, Pia M. et al. 0 0 0 0
0 0 0 Default 0
1 0 US 20030018077 A1 US-PGPUB 20030123
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1 0 US 20030017466 A1 US-PGPUB 20030123
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1 0 US 20020194645 A1 US-PGPUB 20021219
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Yanofsky, Martin F. et al. 0 0 0 0 0 0 0
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1 0 US 20020182698 A1 US-PGPUB 20021205
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536/23.2 O'Malley, Bert W. et al. 0 0 0 0 0
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1 0 US 20020168711 A1 US-PGPUB 20021114
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435/320.1; 435/325; 530/350; 536/23.1 Rosen, Craig A. et al. 0 0
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1 0 US 20020161212 A1 US-PGPUB 20021031 BPC-
1: a secreted brain-specific protein expressed and secreted by prostate and bladder cancer
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Afar, Daniel E. et al. 0 0 0 0 0 0 0 Default
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1 0 US 20020150972 A1 US-PGPUB 20021017
34P3D7: a tissue specific protein highly expressed in prostate cancer
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1 0 US 20020147327 A1 US-PGPUB 20021010
Modified steroid hormones for gene therapy and methods for their use
536/23.5 435/320.1; 435/325; 435/69.1; 530/350 O'Malley, Bert
W. et al. 0 0 0 0 0 0 0 Default 0
1 0 US 20020137209 A1 US-PGPUB 20020926
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1 0 US 20020137019 A1 US-PGPUB 20020926
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Garabedian, Michael et al. 0 0 0 0 0 0 0
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1 0 US 20020132293 A1 US-PGPUB 20020919
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al. 0 0 0 0 0 0 0 Default 0
1 0 US 20020103353 A1 US-PGPUB 20020801
Sequences characteristic of hypoxia-regulated gene transcription
536/23.2 435/320.1; 435/325; 435/69.1 Einat, Paz et al.
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1 0 US 20020068345 A1 US-PGPUB 20020606
98P7C3: homeodomain protein highly expressed in various cancers
435/183 435/320.1; 435/325; 435/69.1 Challita-Eid, Pia M.
et al. 0 0 0 0 0 0 0 Default 0
1 0 US 20020055478 A1 US-PGPUB 20020509 GTP-
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0 Default 0
1 0 US 20020049254 A1 US-PGPUB 20020425
Compounds which interact with the thyroid hormone receptor for the treatment of
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John 0 0 0 0 0 0 0 Default 0
1 0 US 20020039762 A1 US-PGPUB 20020404 26908
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1 0 US 20020012966 A1 US-PGPUB 20020131 18
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1 0 US 20020012961 A1 US-PGPUB 20020131
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0 0 0 0 0 0 Default 0
1 0 US 6645974 B2 USPAT 20031111
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0 0 Default 0
1 0 US 6642362 B1 USPAT 20031104 Genes
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0 0 0 0 0 0 Default 0
1 0 US 6641810 B2 USPAT 20031104
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1 0 US 6599698 B1 USPAT 20030729
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1 0 US 6566078 B1 USPAT 20030520
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0 0 0 0 0 0 0 Default 0
1 0 US 6432920 B1 USPAT 20020813 Nck
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530/326 Sparks, Andrew B. et al. 0 0 0 0 0
0 0 Default 0
1 0 US 6416998 B1 USPAT 20020709
Plasmid encoding a modified steroid hormone 435/325
435/252.3; 435/320.1; 536/23.4; 536/23.5 O'Malley, Bert W. et al.
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1 0 US 6414026 B1 USPAT 20020702
Compounds which interact with the thyroid hormone receptor for the treatment of
fibrotic disease 514/570 514/567 Billingham, Michael
Edward John 0 0 0 0 0 0 0 Default 0
1 0 US 6410245 B1 USPAT 20020625
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coactivator interactions 435/7.1 424/141.1; 435/69.1; 435/7.2;
436/518; 536/23.1; 536/23.5 Northrop, Jeffrey P. et al. 0 0 0
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1 0 US 6387673 B1 USPAT 20020514
Compounds useful for the modulation of processes mediated by nuclear hormone
receptors, methods for the identification and use of such compounds
435/184 435/195; 435/197; 435/7.21; 514/557; 530/350; 530/387.9;

552/653 Evans, Ronald M. et al. 0 0 0 0 0
0 0 Default 0
1 0 US 6384208 B1 USPAT 20020507
Sequence directed DNA binding molecules compositions and methods
536/24.1 536/23.1 Edwards, Cynthia A. et al. 0 0
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1 0 US 6348497 B1 USPAT 20020219
Compounds which interact with the thyroid hormone receptor for the treatment of fibrotic disease 514/570 514/567 Billingham, Michael Edward John 0 0 0 0 0 0 Default 0
1 0 US 6319663 B1 USPAT 20011120
Method for the identification and use of substances that modulate POD function and/or structure 435/4 435/5; 435/6; 435/69.1; 530/350; 536/23.5; 536/23.72 Doucas, Vassilis et al. 0 0 0 0 0 0
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1 0 US 6281330 B1 USPAT 20010828
Multimeric forms of members of the steroid/thyroid superfamily of receptors with the ultraspiracle receptor 530/324 Evans, Ronald M. et al. 0 0 0 0 0 0 0 Default 0
1 0 US 6277972 B1 USPAT 20010821 BPC-1: a secreted brain-specific protein expressed and secreted by prostate and bladder cancer cells 536/23.1 435/320.1; 435/325; 435/69.1 Afar, Daniel E. et al. 0 0 0 0 0 0 0 Default 0
1 0 US 6235496 B1 USPAT 20010522
Nucleic acid encoding mammalian mu opioid receptor 435/69.1
435/252.33; 435/320.1; 435/325; 435/358; 435/365; 536/23.1 Yu, Lei 0 0 0 0 0 0 0 Default 0
1 0 US 6210974 B1 USPAT 20010403
Compositions and methods for promoting nerve regeneration
436/501 436/34; 436/63; 436/86; 436/91 Gold, Bruce G.
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1 0 US 6184205 B1 USPAT 20010206 GRB2 SH3 binding peptides and methods of isolating and using same 514/13
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1 0 US 6103492 A USPAT 20000815
Polynucleotide encoding mu opioid receptor 435/69.1 435/252.3; 435/320.1; 435/325; 435/471; 435/70.1; 435/71.1; 435/71.2; 536/23.5 Yu, Lei 0 0 0 0 0 0 0 Default 0
1 0 US 6010849 A USPAT 20000104
Sequence-directed DNA binding molecules compositions and methods
435/6 435/7.1 Edwards, Cynthia A. et al. 0 0 0
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1 0 US 5968921 A USPAT 19991019
Compositions and methods for promoting nerve regeneration

514/183 514/330; 514/423; 514/428; 514/465; 514/466; 514/534; 514/547; 514/548; 514/549 Gold, Bruce G. 0 0 0 0
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1 0 US 5935934 A USPAT 19990810
Mutated steroid hormone receptors, methods for their use and molecular switch for gene therapy 514/44 424/93.21 Vegeto, Elisabetta et al.
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1 0 US 5874534 A USPAT 19990223
Mutated steroid hormone receptors, methods for their use and molecular switch for gene therapy 530/350 536/23.1; 536/24.1 Vegeto, Elisabetta et al. 0 0 0 0 0 0 0 Default 0
1 0 US 5869241 A USPAT 19990209
Method of determining DNA sequence preference of a DNA-binding molecule
435/6 435/91.1; 435/91.2 Edwards, Cynthia A. et al. 0 0
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1 0 US 5744131 A USPAT 19980428
Sequence-directed DNA-binding molecules compositions and methods
424/78.08 436/501; 514/1 Edwards, Cynthia A. et al. 0
0 0 0 0 0 0 Default 0
1 0 US 5738990 A USPAT 19980414
Sequence-directed DNA-binding molecules compositions and methods
435/6 435/320.1; 435/69.1; 536/24.1 Edwards, Cynthia A. et al.
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1 0 US 5726014 A USPAT 19980310
Screening assay for the detection of DNA-binding molecules 435/6
435/91.2; 436/501 Edwards, Cynthia A. et al. 0 0 0
0 0 0 0 Default 0
1 0 US 5716780 A USPAT 19980210
Method of constructing sequence-specific DNA-binding molecules 435/6
436/501 Edwards, Cynthia A. et al. 0 0 0 0
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1 0 US 5693463 A USPAT 19971202
Method of ordering sequence binding preferences of a DNA-binding molecule
435/6 435/7.23; 536/23.1 Edwards, Cynthia A. et al. 0 0
0 0 0 0 0 Default 0
1 0 US 5639616 A USPAT 19970617
Isolated nucleic acid encoding a ubiquitous nuclear receptor
435/7.1 435/252.3; 435/320.1; 435/69.1; 536/23.5; 536/24.3
Liao, Shutsung et al. 0 0 0 0 0 0 0 Default 0
1 0 US 5578444 A USPAT 19961126
Sequence-directed DNA-binding molecules compositions and methods
435/6 435/7.23; 536/23.1 Edwards, Cynthia A. et al. 0 0
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1 0 US 5565352 A USPAT 19961015
Deubiquitinating enzyme: compositions and methods 435/255.1
435/201; 435/252.3; 435/254.11; 435/254.2; 435/254.21; 435/255.2;
435/320.1; 536/23.2 Hochstrasser, Mark et al. 0 0 0 0
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0 0 US 5556956 A USPAT 19960917 25
Methods and compositions relating to the androgen receptor gene and uses thereof
536/24.1 536/23.1; 536/24.3; 536/24.31 Roy, Arun K. et al.
1 0 0 0 0 0 0 US Full Image US 5556956
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0 0 US 20030175817 A1 US-PGPUB 20030918 13
Method of screening for antiandrogen agent 435/7.2 514/12;
514/169; 530/399; 552/500 Ikeda, Kyoji et al. 1 0 0 0
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1 0 US 20030118997 A1 US-PGPUB 20030626 178 Human
cDNAs and proteins and uses thereof 435/6 435/183; 435/320.1; 435/325;
435/69.1; 536/23.2 Bejanin, Stephane et al. 0 0 0 0
0 0 0 PGPubs Full Image US 20030118997 0
1 0 US 20030022243 A1 US-PGPUB 20030130 48 Protein
aggregation assays and uses thereof 435/7.1 435/7.21
Kondejewski, Les et al. 0 0 0 0 0 0 0
PGPubs Full Image US 20030022243 0
1 0 US 20030017503 A1 US-PGPUB 20030123 14
Methods and labeled molecules for determining ligand binding to steroid
receptors 435/7.1 540/114 Goldrick, Susan E. et al.
0 0 0 0 0 0 0 PGPubs Full Image US
20030017503 0
1 0 US 20030008807 A1 US-PGPUB 20030109 68 Novel
signaling pathway for the production of inflammatory pain and neuropathy 514/1
435/7.21 Levine, Jon David et al. 0 0 0 0
0 0 0 PGPubs Full Image US 20030008807 0
0 0 US 6297013 B1 USPAT 20011002 47
Compositions and methods for determining the activity of DNA-binding proteins
and of initiation of transcription 435/6 435/196; 435/320.1; 435/7.6;
536/23.1; 536/23.2; 536/24.1 Morgan, Antony R. et al. 1 0
0 0 0 0 0 US Full Image US 6297013 0
1 0 US 6291637 B1 USPAT 20010918 41
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critical RNA-protein interactions 530/300 530/323; 530/324; 530/326;
536/23.1; 536/24.1; 536/24.5 Das, Saumitra et al. 0 0 0
0 0 0 0 US Full Image US 6291637 0
1 0 US 6284468 B1 USPAT 20010904 46
Composition and methods for determining the activity of DNA-binding proteins
and of initiation of trascription 435/6 435/196; 435/199; 435/320.1;
536/23.1; 536/24.1 Morgan, Robert Charles et al. 0 0 0 0
0 0 0 US Full Image US 6284468 0
1 0 US 6265213 B1 USPAT 20010724 46
Compositions and methods for determining the activity of DNA-binding proteins
and of initiation of transcription 435/320.1 435/196; 435/6; 530/350;
536/23.1 Morgan, Antony R. et al. 0 0 0 0 0
0 0 US Full Image US 6265213 0
1 0 US 5989904 A USPAT 19991123 37
Selective inhibition of internally initiated RNA translation 435/320.1
435/325; 435/375; 435/455; 435/6; 536/24.1; 536/24.5 Das, Saumitra
et al. 0 0 0 0 0 0 0 US Full Image US 5989904
0

1 0 US 5605929 A USPAT 19970225 65
Methods and compositions for inhibiting 5.alpha.-reductase activity
514/456 514/544; 549/406; 560/70 Liao, Shutsung et al. 0
0 0 0 0 0 0 US Full Image US 5605929 0
1 0 WO 200264017 A DERWENT 20020822 New immortalized human prostatic tumor cell, useful for screening genes indicative or predictive of human cancer, or for testing and screening compounds that alter tumor cell metabolism MOUL, J W et al. 0 0 0
0 0 0 0 Default 0

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 FILE 'MEDLINE, EMBASE, BIOSIS' ENTERED AT 14:38:19 ON 08 JAN 2004
L1         19082 S "ANDROGEN RECEPTOR"
L2            17 S "COREGULATORY PROTEIN"
L3        131322 S "TRANSCRIPTION FACTOR"
L4           622 S L1 AND L3
L5           349 DUP REM L4 (273 DUPLICATES REMOVED)
L6           171 S L5 NOT PY>=2001
L7             8 S "METHOD OF SCREENING" (P) ANDROGEN
L8             0 S ANTIANDROGEN AND L2
L9             0 S L1 AND L2 AND L3
L10            4 S L2 AND ANDROGEN
L11           51 S L1 (P) COFACTOR
L12           23 DUP REM L11 (28 DUPLICATES REMOVED)
L13            9 S L12 NOT PY>=2001
L14            9 S L3 AND L1 AND L11
L15          304 S L1 AND "PROTEIN INTERACTION"
L16          244 DUP REM L15 (60 DUPLICATES REMOVED)
L17           92 S L16 NOT PY>=2001
L18         1573 S L1 AND ANTIANDROGEN
L19            0 S L18 AND "METHOD OF SCREENING"
L20          211 S L18 AND INHIBITOR
L21          950 DUP REM L18 (623 DUPLICATES REMOVED)
L22            2 DUP REM L10 (2 DUPLICATES REMOVED)
L23          151 DUP REM L20 (60 DUPLICATES REMOVED)
L24           98 S L23 NOT PY>=2001
L25           40 S "ANDROGEN-DEPENDENT GENE EXPRESSION"
L26           18 DUP REM L25 (22 DUPLICATES REMOVED)
L27           14 S L26 NOT PY>=2001
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L10 ANSWER 1 OF 4 MEDLINE on STN
ACCESSION NUMBER: 2001038817 MEDLINE
DOCUMENT NUMBER: 20350647 PubMed ID: 10894149
TITLE: PNRC: a proline-rich nuclear receptor **coregulatory protein** that modulates transcriptional activation of multiple nuclear receptors including orphan receptors SF1 (steroidogenic factor 1) and ERRalpha1 (estrogen related receptor alpha-1).
AUTHOR: Zhou D; Quach K M; Yang C; Lee S Y; Pohajdak B; Chen S
CORPORATE SOURCE: Division of Immunology, Beckman Research Institute of the City of Hope, Duarte, California 91010, USA.
CONTRACT NUMBER: CA-44735 (NCI)
SOURCE: MOLECULAR ENDOCRINOLOGY, (2000 Jul) 14 (7) 986-98.
Journal code: 8801431. ISSN: 0888-8809.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200011
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20001124

AB PNRC (proline-rich nuclear receptor **coregulatory protein**) was identified using bovine SF1 (steroidogenic factor 1) as the bait in a yeast two-hybrid screening of a human mammary gland cDNA expression library. PNRC is unique in that it has a molecular mass of 35 kDa, significantly smaller than most of the coregulatory proteins reported so far, and it is proline-rich. PNRC's nuclear localization was demonstrated by immunofluorescence and Western blot analyses. In the yeast two-hybrid assays, PNRC interacted with the orphan receptors SF1 and ERRalpha1 in a ligand-independent manner. PNRC was also found to interact with the ligand-binding domains of all the nuclear receptors tested including estrogen receptor (ER), **androgen** receptor (AR), glucocorticoid receptor (GR), progesterone receptor (PR), thyroid hormone receptor (TR), retinoic acid receptor (RAR), and retinoid X receptor (RXR) in a ligand-dependent manner. Functional AF2 domain is required for nuclear receptors to bind to PNRC. Furthermore, in vitro glutathione-S-transferase pull-down assay was performed to demonstrate a direct contact between PNRC and nuclear receptors such as SF1. Coimmunoprecipitation experiment using Hela cells that express PNRC and ER was performed to confirm the interaction of PNRC and nuclear receptors in vivo in a ligand-dependent manner. PNRC was found to function as a coactivator to enhance the transcriptional activation mediated by SF1, ERR1 (estrogen related receptor alpha-1), PR, and TR. By examining a series of deletion mutants of PNRC using the yeast two-hybrid assay, a 23-amino acid (aa) sequence in the carboxy-terminal region, aa 278-300, was shown to be critical and sufficient for the interaction with nuclear receptors. This region is proline rich and contains a SH3-binding motif, S-D-P-P-S-P-S. Results from the mutagenesis study demonstrated that the two conserved proline (P) residues in this motif are crucial for PNRC to interact with the nuclear receptors. The exact 23-amino acid sequence was also found in another protein isolated from the same yeast two-hybrid screening study. These two proteins belong to a new family of nuclear receptor coregulatory proteins.

L10 ANSWER 2 OF 4 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
ACCESSION NUMBER: 2001125070 EMBASE
TITLE: PNRC: A proline-rich nuclear receptor **coregulatory protein** that modulates transcriptional activation of multiple nuclear receptors including orphan receptors SF1 (steroidogenic factor 1) and ERR.alpha.1 (estrogen related receptor .alpha.-1).

AUTHOR: Zhou D.; Quach K.M.; Yang C.; Lee S.Y.; Pohajdak B.; Chen S.
CORPORATE SOURCE: S. Chen, Division of Immunology, Beckman Coulter, Inc., Res. Institute of the City of Hope, Duarte, CA 91010, United States. schen@coh.org
SOURCE: Molecular Endocrinology, (2000) 14/7 (986-998).
Refs: 42
ISSN: 0888-8809 CODEN: MOENEN
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 003 Endocrinology
016 Cancer
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English

AB PNRC (proline-rich nuclear receptor **coregulatory protein**) was identified using bovine SF1 (steroidogenic factor 1) as the bait in a yeast two-hybrid screening of a human mammary gland cDNA expression library. PNRC is unique in that it has a molecular mass of 35 kDa, significantly smaller than most of the coregulatory proteins reported so far, and it is proline-rich. PNRC's nuclear localization was demonstrated by immunofluorescence and Western blot analyses. In the yeast two-hybrid assays, PNRC interacted with the orphan receptors SF1 and ERR.alpha.1 in a ligand-independent manner. PNRC was also found to interact with the ligand-binding domains of all the nuclear receptors tested including estrogen receptor (ER), **androgen** receptor (AR), glucocorticoid receptor (GR), progesterone receptor (PR), thyroid hormone receptor (TR), retinoic acid receptor (RAR), and retinoid X receptor (RXR) in a ligand-dependent manner. Functional AF2 domain is required for nuclear receptors to bind to PNRC. Furthermore, in vitro glutathione-S-transferase pull-down assay was performed to demonstrate a direct contact between PNRC and nuclear receptors such as SF1. Coimmunoprecipitation experiment using Hela cells that express PNRC and ER was performed to confirm the interaction of PNRC and nuclear receptors in vivo in a ligand-dependent manner. PNRC was found to function as a coactivator to enhance the transcriptional activation mediated by SF1, ERR1 (estrogen related receptor .alpha.-1), PR, and TR. By examining a series of deletion mutants of PNRC using the yeast two-hybrid assay, a 23-amino acid (aa) sequence in the carboxy-terminal region, aa 278-300, was shown to be critical and sufficient for the interaction with nuclear receptors. This region is proline rich and contains a SH3-binding motif, S-D-P-P-S-P-S. Results from the mutagenesis study demonstrated that the two conserved proline (P) residues in this motif are crucial for PNRC to interact with the nuclear receptors. The exact 23-amino acid sequence was also found in another protein isolated from the same yeast two-hybrid screening study. These two proteins belong to a new family of nuclear receptor coregulatory proteins.

L10 ANSWER 3 OF 4 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2001:164301 BIOSIS
DOCUMENT NUMBER: PREV200100164301
TITLE: PNRC: A proline-rich nuclear receptor **coregulatory protein** that modulates transcriptional activation of multiple nuclear receptors including orphan receptors SF1 (steroidogenic factor 1) and ERRalpha1 (estrogen related receptor alpha-1).
AUTHOR(S): Zhou, Dujin; Quach, Keith M.; Yang, Chun; Lee, Stella Y.; Pohajdak, Bill; Chen, Shiuan [Reprint author]
CORPORATE SOURCE: Division of Immunology, Beckman Coulter, Inc. Research Institute of the City of Hope, Duarte, CA, 91010, USA schen@coh.org
SOURCE: Molecular Endocrinology, (July, 2000) Vol. 14, No. 7, pp. 986-998. print.
CODEN: MOENEN. ISSN: 0888-8809.

DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 4 Apr 2001
Last Updated on STN: 15 Feb 2002

AB PNRC (proline-rich nuclear receptor **coregulatory protein**) was identified using bovine SF1 (steroidogenic factor 1) as the bait in a yeast two-hybrid screening of a human mammary gland cDNA expression library. PNRC is unique in that it has a molecular mass of 35 kDa, significantly smaller than most of the coregulatory proteins reported so far, and it is proline-rich. PNRC's nuclear localization was demonstrated by immunofluorescence and Western blot analyses. In the yeast two-hybrid assays, PNRC interacted with the orphan receptors SF1 and ERRalpha1 in a ligand-independent manner. PNRC was also found to interact with the ligand-binding domains of all the nuclear receptors tested including estrogen receptor (ER), **androgen** receptor (AR), glucocorticoid receptor (GR), progesterone receptor (PR), thyroid hormone receptor (TR), retinoic acid receptor (RAR), and retinoid X receptor (RXR) in a ligand-dependent manner. Functional AF2 domain is required for nuclear receptors to bind to PNRC. Furthermore, in vitro glutathione-S-transferase pull-down assay was performed to demonstrate a direct contact between PNRC and nuclear receptors such as SF1. Coimmunoprecipitation experiment using Hela cells that express PNRC and ER was performed to confirm the interaction of PNRC and nuclear receptors in vivo in a ligand-dependent manner. PNRC was found to function as a coactivator to enhance the transcriptional activation mediated by SF1, ERR1 (estrogen related receptor alpha-1), PR, and TR. By examining a series of deletion mutants of PNRC using the yeast two-hybrid assay, a 23-amino acid (aa) sequence in the carboxy-terminal region, aa 278-300, was shown to be critical and sufficient for the interaction with nuclear receptors. This region is proline rich and contains a SH3-binding motif, S-D-P-P-S-P-S. Results from the mutagenesis study demonstrated that the two conserved proline (P) residues in this motif are crucial for PNRC to interact with the nuclear receptors. The exact 23-amino acid sequence was also found in another protein isolated from the same yeast two-hybrid screening study. These two proteins belong to a new family of nuclear receptor coregulatory proteins.

L10 ANSWER 4 OF 4 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2000:468388 BIOSIS
DOCUMENT NUMBER: PREV200000468388
TITLE: Expression of **androgen** receptor coregulatory proteins in prostate cancer and stromal-cell culture models.
AUTHOR(S): Nessler-Menardi, Claudia; Jotova, Iveta; Culig, Zoran; Eder, Iris E.; Putz, Thomas; Bartsch, Georg; Klocker, Helmut [Reprint author]
CORPORATE SOURCE: Department of Urology, University of Innsbruck, Anichstrasse 35, A-6020, Innsbruck, Austria
SOURCE: Prostate, (October 1, 2000) Vol. 45, No. 2, pp. 124-131. print.
CODEN: PRSTDS. ISSN: 0270-4137.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 1 Nov 2000
Last Updated on STN: 10 Jan 2002

AB BACKGROUND: **Androgen** receptor (AR) transcriptional activity is modulated by cofactor proteins. They act as costimulators, corepressors, or bridging proteins, and a disbalanced expression may contribute to the altered activity of the AR in advanced prostate cancer. We investigated the expression of a series of steroid receptor cofactors in prostate cancer cell lines, including several LNCaP sublines, and in prostate stromal cells. METHODS: Expression of cofactors was analyzed by means of RT-PCR in PC-3, Du-145, LNCaP, three sublines of LNCaP established after

long-term **androgen** deprivation, and two strains of primary prostate stroma cells. Expression in LNCaP and LNCaP-abl cells (which represented an advanced tumor cell) was analyzed employing semiquantitative RT-PCR. RESULTS: Ten of the 12 cofactors tested were expressed in all cells analyzed (AIB1, ARA54, ARA70, CBP, cyclin D1, Her2/neu/erbB2, BAG-1/M/L, SRC-1, SMRT, and TIF2). Only ARA55 and FHL2 mRNAs were not detected in all cells. ARA55 mRNA was absent in LNCaP cells, LNCaP sublines, and DU-145 cells; FHL2 was not expressed in LNCaP cells and its derivatives. The expression pattern was identical in LNCaP cells, and the long-term **androgen** ablated LNCaP sublines. Moreover, comparison of expression levels in LNCaP and LNCaP-abl cells revealed a slight reduction in LNCaP-abl cells but no gross differences. CONCLUSIONS: Prostatic cells express a great number of steroid receptor cofactors. AR activity thus seems to be modulated in a very complex way in prostate cells.

13 ANSWER 1 OF 9 MEDLINE on STN
ACCESSION NUMBER: 2001069535 MEDLINE
DOCUMENT NUMBER: 20525399 PubMed ID: 11071847
TITLE: Protein inhibitor of activated STAT3 regulates androgen receptor signaling in prostate carcinoma cells.
AUTHOR: Junicho A; Matsuda T; Yamamoto T; Kishi H; Korkmaz K; Saatcioglu F; Fuse H; Muraguchi A
CORPORATE SOURCE: Department of Urology, Faculty of Medicine, Toyama Medical and Pharmaceutical University, 2630 Sugitani, Toyama 930-0194, Japan.
SOURCE: BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS, (2000 Nov 11) 278 (1) 9-13.
Journal code: 0372516. ISSN: 0006-291X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200101
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20010104

AB Protein inhibitor of activated STAT3 (PIAS3) is a specific inhibitor of signal transducer and activator of transcription 3 (STAT3). PIAS3 binds to STAT3 and inhibits its DNA-binding activity, and thereby STAT3-mediated gene activation. PIAS1, another member of the PIAS family, was recently shown to interact with the **androgen receptor** (AR), a nuclear hormone receptor that has an important role in both physiological and pathological processes, and acts as a **cofactor** for AR. Here we demonstrate that PIAS3 is expressed in prostate cancer cells and its expression is induced in response to dihydrotestosterone (DHT) treatment. Ectopic overexpression of PIAS3 suppressed AR-mediated gene activation induced by DHT-stimulation in LNCaP cells. We provide evidence that these activities were due to direct physical interactions between PIAS3 and AR. These results indicate that PIAS3 acts as a coregulator of AR signaling pathway in prostate cancer cells.

L13 ANSWER 2 OF 9 MEDLINE on STN
ACCESSION NUMBER: 2001022482 MEDLINE
DOCUMENT NUMBER: 20481833 PubMed ID: 11027411
TITLE: Expression of androgen receptor coregulatory proteins in prostate cancer and stromal-cell culture models.
AUTHOR: Nessler-Menardi C; Jotova I; Culig Z; Eder I E; Putz T; Bartsch G; Klocker H
CORPORATE SOURCE: Department of Urology, University of Innsbruck, Innsbruck, Austria.
SOURCE: PROSTATE, (2000 Oct 1) 45 (2) 124-31.
Journal code: 8101368. ISSN: 0270-4137.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200011
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20001109

AB BACKGROUND: **Androgen receptor** (AR) transcriptional activity is modulated by **cofactor** proteins. They act as costimulators, corepressors, or bridging proteins, and a disbalanced expression may contribute to the altered activity of the AR in advanced prostate cancer. We investigated the expression of a series of steroid receptor cofactors in prostate cancer cell lines, including several LNCaP sublines, and in prostate stromal cells. METHODS: Expression of cofactors

was analyzed by means of RT-PCR in PC-3, Du-145, LNCaP, three sublines of LNCaP established after long-term androgen deprivation, and two strains of primary prostate stroma cells. Expression in LNCaP and LNCaP-abl cells (which represented an advanced tumor cell) was analyzed employing semiquantitative RT-PCR. RESULTS: Ten of the 12 cofactors tested were expressed in all cells analyzed (AIB1, ARA54, ARA70, CBP, cyclin D1, Her2/neu/erbB2, BAG-1/M/L, SRC-1, SMRT, and TIF2). Only ARA55 and FHL2 mRNAs were not detected in all cells. ARA55 mRNA was absent in LNCaP cells, LNCaP sublines, and DU-145 cells; FHL2 was not expressed in LNCaP cells and its derivatives. The expression pattern was identical in LNCaP cells, and the long-term androgen ablated LNCaP sublines. Moreover, comparison of expression levels in LNCaP and LNCaP-abl cells revealed a slight reduction in LNCaP-abl cells but no gross differences. CONCLUSIONS: Prostatic cells express a great number of steroid receptor cofactors. AR activity thus seems to be modulated in a very complex way in prostate cells.

L13 ANSWER 3 OF 9 MEDLINE on STN
ACCESSION NUMBER: 2000120800 MEDLINE
DOCUMENT NUMBER: 20120800 PubMed ID: 10654935
TITLE: FHL2, a novel tissue-specific coactivator of the androgen receptor.
AUTHOR: Muller J M; Isele U; Metzger E; Rempel A; Moser M; Pscherer A; Breyer T; Holubarsch C; Buettner R; Schule R
CORPORATE SOURCE: Universitats-Frauenklinik, Abteilung Frauenheilkunde und Geburtshilfe I, Klinikum der Universitat Freiburg, Breisacherstrasse 117, 79106 Freiburg, Germany.
SOURCE: EMBO JOURNAL, (2000 Feb 1) 19 (3) 359-69.
Journal code: 8208664. ISSN: 0261-4189.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200003
ENTRY DATE: Entered STN: 20000327
Last Updated on STN: 20000327
Entered Medline: 20000310

AB The control of target gene expression by nuclear receptors requires the recruitment of multiple cofactors. However, the exact mechanisms by which nuclear receptor-**cofactor** interactions result in tissue-specific gene regulation are unclear. Here we characterize a novel tissue-specific coactivator for the **androgen receptor** (AR), which is identical to a previously reported protein FHL2/DRAL with unknown function. In the adult, FHL2 is expressed in the myocardium of the heart and in the epithelial cells of the prostate, where it colocalizes with the AR in the nucleus. FHL2 contains a strong, autonomous transactivation function and binds specifically to the AR in vitro and in vivo. In an agonist- and AF-2-dependent manner FHL2 selectively increases the transcriptional activity of the AR, but not that of any other nuclear receptor. In addition, the transcription of the prostate-specific AR target gene probasin is coactivated by FHL2. Taken together, our data demonstrate that FHL2 is the first LIM-only coactivator of the AR with a unique tissue-specific expression pattern.

L13 ANSWER 4 OF 9 MEDLINE on STN
ACCESSION NUMBER: 1999333911 MEDLINE
DOCUMENT NUMBER: 99333911 PubMed ID: 10405524
TITLE: Differential induction of the androgen receptor transcriptional activity by selective androgen receptor coactivators.
AUTHOR: Yeh S; Chang H C; Miyamoto H; Takatera H; Rahman M; Kang H Y; Thin T H; Lin H K; Chang C

CORPORATE SOURCE: George Whipple Laboratory for Cancer Research, Department of Pathology, University of Rochester, NY 14642, USA.
SOURCE: KEIO JOURNAL OF MEDICINE, (1999 Jun) 48 (2) 87-92. Ref: 40
Journal code: 0376354. ISSN: 0022-9717.
PUB. COUNTRY: Japan
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199908
ENTRY DATE: Entered STN: 19990820
Last Updated on STN: 19990820
Entered Medline: 19990811

AB Several new **androgen receptor** (AR) cofactors, associated to the ligand binding domain of AR, have been identified by our group and named AR associated protein (ARA)70, ARA55, and ARA54. Our previous reports have suggested that the **cofactor** ARA70 can confer the androgenic effect from 17 beta-estradiol (E2) and antiandrogen to AR. It is of interest for us to compare and determine if the specificity of sex hormones and antiandrogens could be modulated by different coactivators. Our results indicate that ARA70 is the best coactivator to confer the androgenic activity on E2. Only ARA70 and ARA55 could increase significantly the androgenic activity of hydroxyflutamide, a widely used antiandrogen for the treatment of prostate cancer. Furthermore, as compared to the relative specificity of these coactivators to AR in the prostate cancer DU145 cells, our results suggest that ARA70 has a relatively higher specificity. Together, our data suggest that the specificity of sex hormones and antiandrogens can be modulated by some selective AR coactivators. These findings may not only help us to better understand the specificity of the sex hormones and antiandrogens, but also to facilitate the development of better antiandrogens or androgens to fight the androgen-related diseases, such as prostate cancer.

L13 ANSWER 5 OF 9 MEDLINE on STN
ACCESSION NUMBER: 97037570 MEDLINE
DOCUMENT NUMBER: 97037570 PubMed ID: 8883217
TITLE: Mammalian 3 alpha-hydroxysteroid dehydrogenases.
COMMENT: Erratum in: Steroids 1997 May;62(5):455-6
AUTHOR: Penning T M; Pawlowski J E; Schlegel B P; Jez J M; Lin H K; Hoog S S; Bennett M J; Lewis M
CORPORATE SOURCE: Department of Pharmacology, University of Pennsylvania School of Medicine, Philadelphia 19104-6084, USA.
SOURCE: STEROIDS, (1996 Sep) 61 (9) 508-23. Ref: 64
Journal code: 0404536. ISSN: 0039-128X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199701
ENTRY DATE: Entered STN: 19970219
Last Updated on STN: 20030318
Entered Medline: 19970123

AB Mammalian 3 alpha-hydroxysteroid dehydrogenases (3 alpha-HSDs) regulate steroid hormone levels. For example, hepatic 3 alpha-HSDs inactivate circulating androgens, progestins, and glucocorticoids. In target tissues they regulate access of steroid hormones to steroid hormone receptors. For example, in the prostate 3 alpha-HSD acts as a molecular switch and controls the amount of 5 alpha-dihydrotestosterone that can bind to the **androgen receptor**, while in the brain 3 alpha-HSD can regulate the amount of tetrahydrosteroids that can alter GABAa receptor

function. Molecular cloning indicates that these mammalian 3 alpha-HSDs belong to the aldo-keto reductase superfamily and that they are highly homologous proteins. Using the three-dimensional structure of rat liver 3 alpha-HSD as a template for site-directed mutagenesis, details regarding structure function relationships, including catalysis and **cofactor** and steroid hormone recognition have been elucidated. These details may be relevant to all mammalian 3 alpha-HSDs.

L13 ANSWER 6 OF 9 MEDLINE on STN
ACCESSION NUMBER: 96238290 MEDLINE
DOCUMENT NUMBER: 96238290 PubMed ID: 8787343
TITLE: [5-alpha-reductases: physiology and pathology].
Les 5 alpha-reductases: physiologie et pathologie.
AUTHOR: Mowszowicz I; Berthauit I; Mestayer C; Wright F; Kuttenn F; Mauvais-Jarvis P
CORPORATE SOURCE: Laboratoire de Biochimie, Hopital Necker, Paris.
SOURCE: ANNALES D ENDOCRINOLOGIE, (1995) 56 (6) 555-9. Ref: 27
Journal code: 0116744. ISSN: 0003-4266.
PUB. COUNTRY: France
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: French
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199609
ENTRY DATE: Entered STN: 19961008
Last Updated on STN: 19961008
Entered Medline: 19960926

AB In most androgen target tissues, the first step of androgen action is the 5 alpha-reduction of testosterone to DHT which binds to the **androgen receptor** with an affinity 3 to 4 fold higher than testosterone. Two genes, encoding two isozymes of 5 alpha-reductase (5 alpha-R) have been cloned. The two isoforms, 5 alpha-R1 and 5 alpha-R 2 are located on chromosomes 5 and 2 respectively and differ in optimal pH, substrate and inhibitor affinities and tissue expression. 5 alpha-R 2 is responsible for sexual differentiation. It is the major form expressed in the prostate where it seems necessary for embryonic growth and development. 5 alpha-reductase deficiency results in androgen insensitivity due to abnormal 5 alpha-R 2. Affected patients are XY individuals with a very peculiar form of male pseudohermaphroditism: they have feminine genitalia at birth and masculinize at puberty. 29 mutations, spanning the whole coding portion of the gene, have been described; correlation between mutations and enzyme activity have led to the suggestion that both the N- and the C-terminal end of the gene are involved in substrate binding, whereas the **cofactor** binding-site is located in the C-terminus. In contrast to androgen insensitivity due to 5 alpha-reductase deficiency, increased 5 alpha-reductase activity can result in androgen hypersensitivity as described in idiopathic hirsutism or benign prostatic hyperplasia. In these case 5 alpha-R 1 could possibly be involved.

L13 ANSWER 7 OF 9 MEDLINE on STN
ACCESSION NUMBER: 94000122 MEDLINE
DOCUMENT NUMBER: 94000122 PubMed ID: 8397593
TITLE: [Testosterone versus dihydrotestosterone effects on permanent squamous epithelial cancer cell lines of the larynx].
Testosteron- versus Dihydrotestosteron-Effekte auf permanente Plattenepithelkarzinomzellinien des Larynx.
AUTHOR: Kleemann D
CORPORATE SOURCE: Hals-Nasen-Ohren-Klinik und Poliklinik, Otto Korner, Medizinische Fakultat, Universitat Rostock.
SOURCE: LARYNGO- RHINO- OTOLOGIE, (1993 Aug) 72 (8) 402-5.

Journal code: 8912371. ISSN: 0935-8943.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: German
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199311
ENTRY DATE: Entered STN: 19940117
Last Updated on STN: 19970203
Entered Medline: 19931115

AB Whereas stimulating effects of androgenic hormones on the laryngeal mucosa and their tumours have been reported in the literature, we are faced with the highest incidence of laryngeal carcinomas in the presence of a reduced androgen signal from the testes associated with aging. The discrepancies between reports in the literature and our own recent experiences with in vitro application of testosterone on permanent laryngeal squamous carcinoma cell lines, initiated this current examination of testosterone, dihydrotestosterone (DHT) and cyproterone acetate effects on two different laryngeal cancer cell lines. No DHT and cyproterone acetate effects on the **androgen receptor** negative line UM-SCC11B were found. However, growth of the HEp-2 line was significantly inhibited undergoing the cyproterone acetate application and significantly enhanced after DHT application. Both lines underwent a dose-dependent growth inhibition after testosterone application. These effects seem to be cytostatic rather than cytotoxic. The mechanisms leading to these effects can only be discussed hypothetically at present. Furthermore, if one takes into consideration the decrease of serum testosterone levels in aging males and the near normal levels of DHT in serum and tissues, so one may assume an imbalance between testosterone and DHT as an important **cofactor** in the genesis of laryngeal cancer. Current research knowledge on the basics of benign prostate hyperplasia, several experimental and clinical reports in the ENT literature together with our own experimental results, are leading to a new and hopeful therapeutic opportunity for the future, involving the blocking of 5 alpha reductase as the enzyme which manages the DHT formation from testosterone. (ABSTRACT TRUNCATED AT 250 WORDS)

L13 ANSWER 8 OF 9 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
ACCESSION NUMBER: 94229603 EMBASE
DOCUMENT NUMBER: 1994229603
TITLE: [5.alpha.-Reductases = physiology and pathology]. 5.alpha.-REDUCTASES: PHYSIOLOGIE ET PATHOLOGIE.
AUTHOR: Mowszowicz I.; Berthaut I.; Mestayer C.; Wright F.; Kuttenn F.; Mauvais-Jarvis P.
CORPORATE SOURCE: Laboratoire de Biochimie B, Fac. de Medecine Pitie-Salpetriere,Paris, France
SOURCE: Andrologie, (1994) 4/1 (71-77). ISSN: 1166-2654 CODEN: AROLEO
COUNTRY: France
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 005 General Pathology and Pathological Anatomy
022 Human Genetics
028 Urology and Nephrology
029 Clinical Biochemistry
LANGUAGE: French
SUMMARY LANGUAGE: English; French

AB In most androgen target tissues, the first step of androgen action is the 5.alpha.-reduction of testosterone to DHT which binds to the **androgen receptor** with an affinity 3 to 4 fold higher than testosterone. Two genes, encoding two isozymes of 5.alpha.-reductase (5.alpha.-R) have been cloned. The two isoforms, 5.alpha.-R1 and 5.alpha.-R2 are located on chromosome 5 and 2 respectively and differ in optimal pH, substrate and inhibitor affinities and tissue expression.

5.alpha.-R2 is responsible for sexual differentiation. It is the major form expressed in the prostate where it seems necessary for embryonic growth and development. In this tissue, as in human skin, 5.alpha.-R2 is stimulated by androgens thus amplifying androgen action. 5.alpha.-reductase deficiency results in androgen insensitivity due to abnormal 5.alpha.-R2. Affected patients are XY individuals with a very peculiar form of male pseudohermaphroditism: they have feminine genitalia at birth and masculinize at puberty. Different mutations, spanning the whole coding portion of the gene, have been described; correlation between mutations and enzyme activity have led to the tentative localization of the substrate binding site in exon 1 and the **cofactor** binding site in exon 4. In contrast to androgen insensitivity due to 5.alpha.-reductase deficiency, increased 5.alpha.-reductase activity can result in androgen hypersensitivity as described in idiopathic hirsutism or benign prostatic hyperplasia. In these case antiandrogen therapy, using 5.alpha.-reductase inhibitors, can be considered.

L13 ANSWER 9 OF 9 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1998:175549 BIOSIS
DOCUMENT NUMBER: PREV199800175549
TITLE: CREB-binding protein in androgen receptor-mediated signaling.
AUTHOR(S): Aarnisalo, Piia [Reprint author]; Palvimo, Jorma J.; Janne, Olli A.
CORPORATE SOURCE: Inst. Biomed., Dep. Physiol., P.O. Box 9, Univ. Helsinki, FIN-00014 Helsinki, Finland
SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (March 3, 1998) Vol. 95, No. 5, pp. 2122-2127. print.
CODEN: PNASA6. ISSN: 0027-8424.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 20 Apr 1998
Last Updated on STN: 20 Apr 1998

AB CREB-binding protein (CBP) is a transcriptional coregulator that interacts with different DNA binding proteins and components of the general transcription machinery. CBP enhanced androgen receptor (AR)-dependent transcription under transient transfection conditions in CV-1 cells. The ligand binding domain (LBD) and residues 38-296 of the N-terminal region of AR are not required because the activity of a receptor mutant devoid of these domains was augmented by coexpressed CBP. There is physical interaction between AR and CBP in vivo, as judged by coimmunoprecipitation experiments from cell extracts. Consistent with the role of CBP as a coactivator for AR, the 12S E1A adenoviral protein that inactivates CBP function strongly inhibited AR-dependent transactivation. Exogenous CBP was also capable of overcoming the inhibitory effect of AR on AP-1 activity and diminished the mutual transcriptional repression between AR and NF-kappaB (RetA). Collectively, these data imply that transcriptional interference between AR and AP-1 or NF-kappaB is mediated, at least in part, through competition for intracellular CBP and that this coactivator serves as an integrator between androgen-mediated and other signaling pathways.

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